

Identification of phenotypic defects in the zinc finger transcription factor *ztf-29*

Trae Dunn,¹ Justice Brakache¹, Kerry McCardel¹, Ciara Hosea^{1,2}, and Martin. L Hudson¹

1. Department of Cellular and Molecular Biology, Kennesaw State University
2. Molecular Virology and Microbiology, Baylor College of Medicine

Aging and many aging-associated diseases such as Alzheimer's disease and cancer are characterized by a progressive decline in physiological functions and a decline in the ability to respond to stress. The underlying causes for many aging-associated diseases are unknown. Identifying genes that control normal aging will advance our understanding of the molecular changes that underlie the aging process and might help treat age-associated diseases. In order to get a better understanding of the molecular mechanisms behind these diseases, we utilize the nematode *Caenorhabditis elegans* as a model organism to examine defects in physiology and aging.

The human PRDM genes code for zinc finger transcription factors, and mutations in these genes are associated with mixed lineage leukemia and other cancers. To gain a better understanding of how PRDM genes work at the molecular level and how they interact with other cancer and aging related genes, we examined a mutation in *ztf-29*, which is the *C. elegans* ortholog of PRDM16. Using a *kal-1-GFP* reporter gene, we found that *ztf-29* mutants exhibit defects in head neuron positioning, including localization and morphology. We also analyzed *ztf-29* mutants for behavioral phenotypes using video tracking, which revealed defects in locomotion including a significant reduction in the average number of reversals performed. Finally, we used a mitochondrial stress reporter gene to determine if *ztf-29* has roles in mitochondrial homeostasis. We find that *ztf-29* mutants exhibit chronic mitochondrial stress responses, suggesting that *ztf-29* may have a role in mitochondrial gene regulation and/or function. Based on these results, we conclude that *ztf-29* plays a significant role in *C. elegans* nervous system development along with the maintenance of mitochondrial physiology. Work is on-going to rescue *ztf-29* mutant phenotypes and to gain a better understanding of what other genes *ztf-29* might be working with during normal development and physiology.